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Electronic Control Devices and the Clinical Milieu

In a 2006 issue of the *Journal,* Nanthakumar et al. (1) presented a study of Taser ECDs (electronic control devices) using 6 pigs averaging 50 kg (110 lb). Research on these devices is important as they are rapidly changing the practices of law enforcement in North America and the United Kingdom with over 620 uses per day. The ECD delivers its short, motor-neuron capturing pulses through a pair of small darts (or barbs) attached by very fine wires. Darts were positioned subcutaneously to maximize current through the heart, in a vector not possible in humans due to the differences in thoracic geometry. Some ventricular capture was seen but no arrhythmias induced during 94 full-strength applications. This is impressive as swine are more sensitive to electrical induction of arrhythmias than are other mammals (2), possibly due to the transmural penetration of the Purkinje fibers (3).

The researchers then infused epinephrine and delivered 16 ECD applications—again in the worst-case position. They had one case of ventricular fibrillation (VF) from these 16 applications. The investigators concluded that these devices may have cardiac risks. Although we recognize the solid reputations of the researchers and the quality review process of *JACC,* we have concerns about the applicability of this conclusion to the clinical setting. The first concern lies with the implicit assumption that the rhythm with a police "in-custody death" is VF. The ECDs are involved in 30% to 32% of in-custody deaths (J. Ho, unpublished data, 2006) (4,5). However, studies have not reported a single case in which the presenting rhythm was VF when an ECD was used. A study of 162 consecutive in-custody deaths found that, whereas there was a significant association of impact weapons with sudden death, the ECDs were never (0 of 50; $p = 0.001$) associated with a sudden collapse. This would seem to eliminate electrically induced VF as the cause of death.

The one anecdote, cited in Nanthakumar et al. (1), of possible electrically induced VF was misreported with material omissions (6). A violent subject exhibiting all the signs of excited delirium was briefly subdued with a short ECD discharge. Paramedics were present and found a normal pulse and respiration *after* the ECD discharge. After a 14-min delay, the subject collapsed and probably had an ideoventricular rhythm. After an aggressive therapy of 3 defibrillation shocks along with atropine and epinephrine, the subject finally had the VF strip shown in the published anecdote. A total of 23 min elapsed between the ECD application and the published VF strip.

Our second concern has to do with the timing of the shocks with the epinephrine infusion. The study does not mention any delay between the time of infusion and the ECD application.

Although perhaps counterintuitive, epinephrine reduces the VF threshold only for the first few minutes (7). After that there is a supratachyphylaxis, and the VF threshold is increased significantly *above* the baseline. This timing is concordant with the clinical scenario. Typically, an individual exhibits violent agitation with hyperactivity (8) for several minutes, and third parties call for help. The police require 5 to 15 min to arrive before they can apply any restraint device. At this point, the adrenergic tone has been elevated for several minutes. An epinephrine infusion minutes before an ECD shock would appear to give results *opposite* of those seen with the clinical scenario.

Finally, we would draw attention to the results of the Cleveland Clinic study published in the same issue of the *Journal* (9). This study used significantly higher exposures (total shock charge of approximately 2,000 five-second weapon discharges per pig versus about 50 for the Nanthakumar et al. [1]) and also evaluated the risk of the induction of ventricular arrhythmias. The investigators found no induction of arrhythmias except at a high multiple of the device output and that cocaine increased this safety margin even further. That would appear to be more consistent with the clinical results in which no objectively documented VF has occurred in 610,000 police uses of these devices.

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Reply

We thank Drs. Pippin and Kroll et al. for their interest in our work (1). We disagree with the notion that experimental models are never of any use in understanding and in studying arrhythmias. Most of the work on mechanisms of fibrillation and defibrillation, including studies in cardiac arrest, have been conducted in guinea pigs, rats, pigs, and dogs. Readers of *JACC* are quite aware of the